PROC. ENTOMOL. SOC. WASH.
92(3), 1990, pp. 373–384

LIKELY CAUSES AND EXPLANATION OF PROBABLE ATAVISM IN A SOMATICALLY MOSAIC FLY FROM A WILD POPULATION (DIPTERA, ASILIDAE, *NANNOCYRTOPOGON MINUTUS*)

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Abstract.—Description is given of an otherwise normal male asilid (of a genus of 28 species having either hyaline or lightly infuscated wings) with its left wing strikingly color-patterned. This appears to be the first recorded not-gynandromorphic, not-parasitized somatic mosaic in Diptera apart from laboratory cultures and experiments. Possible genetic origins of such mosaicism, of phenotypic expression, and their consequences are outlined. Despite lack of relevant fossils, the more plausible conclusion is that the wing pattern is primarily atavistic and not a neomorphism. Mutants calling forth ancestral attributes do not differ qualitatively from those altering familiar, "lesser" phenotypes. Ancestral phenotypic attributes probably regularly disappear long before their genetic mechanisms pass beyond the capacity for reexpression, as substantiated by disappearance and reoccurrence of R_3 in Brachycera.

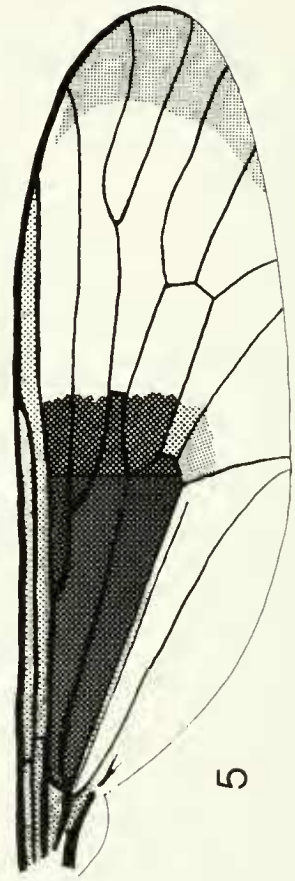
Key Words: Atavism, somatic mosaicism, genetics, wing maculation, wing venation

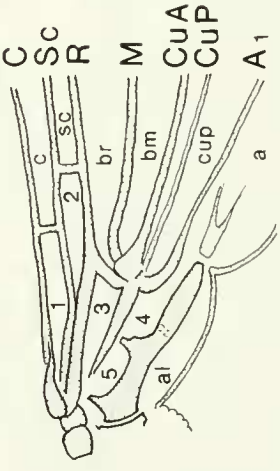
What is to be made of a chimaeric male robber fly, *Nannocyrtopogon minutus* Wilcox and Martin, otherwise normal, having the blade of one wing palely infuscate and normal for the species, the other wing displaying a striking color pattern that is unusual even among asilid species normally having maculated wings (Figs. 1–3, 5)? This in a genus in which the 28 other species do not have color patterns on their wings, being nearly equally divided between those with hyaline and those with lightly tinted wing membranes. The explanation must largely be genetic.

Because genetic systems are subject to mutation, errors of mitosis and fertilization, individuals of a population may be viewed as having their bodies potentially subjected to partition into two or more genetically different sectors during development. In most species, individuals are regarded as "normal" if no disparate sector is detected. Those having from more than 0 to as much as 50% included in such sectors are termed *mosaics*.

Though ordinarily rare, the commonest mosaic detected in wild populations of insects is the gynandromorph, in which the body is partitioned into genetically and phenotypically sexual sectors. Such sexual mosaics, not to be confused with intersexes which are of uniform genotype, have been found in many orders of insects and in many families of flies, though not in the Asilidae.[1]

[1] No museum dipterist of whom I inquired could recollect having read of or seen any mosaic *asilid* (either gynandromorphic or not-gynandromorphic). It is unlikely that striking anomalies of asilids go unnoticed (e.g. see Weinberg 1973). Yet no asilid gynandromorph or other mosaic is recorded by either Zoological Record or Entomological Abstracts (to Volume 20(5), June 1989) within the years they cover for the interval 1925–1989, nor did Collin (1927) mention any earlier records in his brief review.





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Mosaics of a single sex, with an aspect sufficiently striking to be noticed, are well-known but not common in Lepidoptera (Cockayne 1924, Robinson 1971) where striking individual variations in color patterns of wings rarely escape notice. Elsewhere among insects, as with flies in wild populations, they appear to be of a second order of rarity. Apart from certain Nematocera infested with parasites, those recorded for Diptera, of which I am aware, are derived from laboratory cultures and experiments. The earliest general account is that by Morgan and Bridges (1919) for *Drosophila.*

It is assumed that the genetics of asilids, like that of most functionally diploid insects which have been studied, does not depart in any general or unique way from that of species of *Drosophila.* The phenotypic effects of asterisked mutant alleles of *D. melanogaster* Meigen mentioned in discussion (e.g. *Lyra), unless another reference is given, will be found in Lindsley and Grell (1963).

There are accordingly two probable answers to the large question posed by the mosaic asilid, each with more than one possible explanation. They are: the left wing of the mosaic fly may provide a preview of a remarkable apomorphy potentially realizable in the future, or it may display in near entirety a purely atavistic trait.

The Capture

The male mosaic was one of a total of 5 males and 2 females of *N. minutus* collected on July 20 and 26, 1988; the second search was made with the hope that others would be found, perhaps with both wings maculated. The site is approximately 5.2 km northwest of Fawnskin, San Bernardino Co., Ca., at an altitude of roughly 1890 m, not far from some of the formerly recorded sites at which *N. minutus* has been collected. The species is probably generally distributed in the San Gabriel and San Bernardino Mountains (Wilcox and Martin 1957).

At mid-day *N. minutus* sallied after smaller flies from perches on boulders, 30–50 cm in diameter, in the dry bed of Holcomb Creek. Most flies were old, to judge from the torn hind margins of the wings and broken or missing macrochaetae. Oddly, they were found only along a particular length of the creek bed, some 30–40 m long. The total number of individuals along that stretch was almost certainly fewer than two dozen, but more than twelve.

Normal Wing Coloration and Structure

Because the left wing of the mosaic is strikingly unusual by having a color pattern, it was necessary to determine whether it also differs in less obvious features. Though Wilcox and Martin's (1936b) description of *N. minutus* portrays the overall appearance of the fly, the account of the wing is not adequate for close comparison. The following condensed description is drawn from wings of the six normal individuals collected at Holcomb Creek, ten from the University of California collection at Riverside, and from

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Figs. 1–5. Figs. 1–3. Mosaic male of *Nannocyrtopogon minutus* Wilcox and Martin, ca. 13× magnification (actual wing length 4.2 mm). 1—Left side, 2—dorsal aspect, 3—tilted to display right wing. Fig. 4. Diagram of flattened wing base—the proximal portion of radius has folded over the basal half of the basisubcostal cell, 2. Veins: C—costa, Sc—subcosta, R—radius, M—media, CuA—anterior cubitus, CuP—posterior cubitus, A—first anal vein. Lettered cells: c—costal, sc—subcostal, br—basiradial, bm—basimedial, cup—posterior cubital, a—anal. Numbered cells and cell-like enclosures at wing base: 1—basicostal, 2—basisubcostal, 3—first basimedial, stem or prearculus cell, 4—basianal cell, anterior to distal limb of 3rd axillary sclerite, 5—"cell" anterior to proximal limb of 3rd axillary sclerite (the only hyaline cell at the base of the wing). Other: al—alula. Fig. 5. Diagram of patterned areas and venation of left wing of mosaic male (cf. Fig. 1) roughly portraying depths of coloring and extent of pattern—see description for details of pattern.

the right wing of the chimaera. Vein color was viewed by reflected light, wing membrane color by transmitted light. The smallest details mentioned, and common to all, were determined at a magnification of 50×, and checked at 250× in two wings softened with KOH and mounted in euparal.[2] Where possible, nomenclature of veins and cells (Fig. 4) follows McAlpine (1981).

As usual, fluting of wing along longitudinal veins pronounced; veins dark sepia, somewhat lighter as they thin distally; vein MA (arculus), crossvein sc-r, veins CuP and A_2 weakly developed; membrane hyaline throughout, coloring microtrichial (properly acanthal, type b [Richards 1979]); cell c subhyaline to very pale fuscous; cells bc, bsc, stem cell [Shannon's (1924) "prearculus cell"], extreme base of cell a_1, pale to light brown; "cell" bounded above by proximal stem of MP to A_1-complex, and anterior basal portion and proximal lateral apophysis of third axillary sclerite, hyaline (Fig. 4, "cell" 5); cell sc light brown in apical half; short, pale brown streak proximally between veins CuA and CuP in some; a broad, bare hyaline band along length of posterior margin of CuP; remaining membranous areas of wing very light to pale brown, gradually paler posteriorly and basally; alula in part very pale brown, or not (Fig. 4, a1).

The Mosaic

Apart from the surprising 3-partite coloration of 3 grades of saturation marking the blade of the left wing (Figs. 1, 2, 5), a slightly more exaggerated fluting along its longitudinal veins, and minor defects to be discussed, the mosaic specimen is a normal male. In appearance it corresponds well with Wilcox and Martin's description. External morphology of head, antennae,[3] thorax, abdomen, terminalia, legs, patterns of pruinosity and setation, right wing coloration, sizes and venational patterns of both wings (cf. Figs. 1, 3), and body coloring—even at the regions of the thorax bearing the wings—are typical of *N. minutus*.

Left wing stalk, venation, color of the five small "cells" at the stalk of the wing, and general light to pale acanthal browning of all unaffected areas of the wing blade normal for the species. Both membrane and acanthae are colored brown in sharply delimited regions of the blade, giving a much darker, large basal area and a smaller, much less dark one apically, separated by a continuous broad region of a normal light to pale tint (Figs. 1, 2, 5). Thus: cell c pale brown, lighter distally; cell sc light brown from origin to apex, cell br dark brown, somewhat lighter along anterior half; cell bm dark brown, narrowly paler in proximal third along vein CuA; bases of cells r_1 and r_{2+3} dark brown nearly to a line connecting the distal end of vein Sc to crossvein r-m; r_1 lighter along veins R_1 and R_{4+5}; nearly basal 0.4 of cell d (to a point below cross vein r-m) dark brown, darkest basally and along veins M_{1+2} and M_3; basal 0.3+ of cell m_3 and basal third of CuA successively lighter brown. Except at apex, remainder of blade very light to pale brown as in the unaffected right wing. Basal half of wing therefore presents a strongly contrasting dark brown macula in the shape of a slightly opened fan, given added emphasis by the fluting of the wing along the longitudinal veins.

Wing tip with a sharply bounded apical lunule, extending from near apex of cell r_1 to near midpoint of outer margin of cell m_2; greatest width at cell R_4 nearly one-eighth length of wing; much paler than most of

[2] At 250×, slide preparations show an approximately 12-partite internal "annulation" of MP, or bulla, immediately before its bifurcation, without a thyridial clear spot, that is not ordinarily detectable at 50× in pinned specimens. Campaniform sensilla occur along the basal margin of the tegula (*ca.* 18), at base of Sc (20±) and adjacent to its junction with crossvein h (8–9), at base of vein R (*ca.* 70) and widely scattered along its length (6–8) just before and following separation of Rs.

[3] Broken off during photography.

basal "fan," similar in lightness to coloration at base of cell cua only; everywhere contrasting strongly with the lightly tinted adjacent membrane (Figs. 1, 2, 5); alula palely infuscate.

Developmental Defects of the Maculated Wing

The left wing's length (corrected for curvature), veins, venational pattern of cells, and outlines of the wing's margin are all normal. Apart from a slight positive curvature of the blade, physical abnormalities occur in pigmented areas only and appear minor; indeed, detectable only at higher magnifications. At 25×, seven tiny dorsal blisters are visible: two near the distal end of cell bm (70 and 40 μm in greatest diameters), and five in the lunule: one in cell r_3 (20 μm); one in r_4 (30 μm); and three in m_1 (10–20 μm). At 50×, the dorsal membranes over the maculae seem very slightly thicker than the surrounding not-maculated membrane.

These abnormalities are explicable as results of a slight but consistent incoordination during the terminal stages of dorsoventral epithelial contraction of the pupal wing. Their minor nature contrasts markedly with the often extreme abnormalities to be seen among the phenotypes of mutant genes affecting the wings of *Drosophila* (see Waddington 1940, 1942).

Asilid Wing Patterns

Most asilid wings derive their coloration, when present, from type B acanthae, from membrane pigmentation, or from both; rarely is it structural. The wings of the majority of Nearctic asilids range from hyaline through tinged to full color (usually browns to nearly black), or have a gradually deepening color along an axis. A minority have discrete, maculated patterns. The commonest of these is a slight clouding or spotting at crossveins and venational branchpoints. Somewhat less frequent, but widespread, is a darkening of the wing adjacent to the apex, often in the form of a lunule.

Nearly the full range of wing coloration is shown by Nearctic species of *Cyrtopogon,* of which there are some seventy, and from which *Nannocyrtopogon* was split by Wilcox and Martin (1936a). Some, as *C. dasyllis* Williston, *C. maculipennis* (Macquart), and the male of *C. bimacula* (Walker), have large, striking patterns, but in each the principal macula is not proximal, nor is the apex maculate. As Dr. Eric Fisher pointed out to me, however, at least three Palearctic species of *Crytopogon* do have a truly apical macula separated by clear membrane from a more basal pattern; e.g. *C. centralis* Loew, the most similar of these (see Engle 1929, fig. 222, p. 355). Nevertheless, the basal macula of *C. centralis* (apically very similar in outline and extent to that of the mosaic) does not reach the base of the wing, nor is the apical macula a lunule. Though wing patterns of some *Cyrtopogon* seem not far removed, none is wholly like that of the left wing of *N. minutus.* Indeed, none of the species of the other forty-six genera of Nearctic Dasypogoninae[4] have a compound basal and apical pattern closely similar to that of the chimaera, nor do the remaining Nearctic asilids. How then is the occurrence of the two differently colored wings of the aberrant male fly to be explained, and how may the uniqueness of the patterned wing be understood?

Interpretation

Because the patterned wing of the male chimaera is free of striking abnormality in form, basal coloring, veins, and venational pattern, it is unlikely to have been the direct result of an asymmetrically directed envi-

[4] The Dasypogoninae of Martin's and Wilcox' (1965) classification have been split into three allied subfamilies by both Papavero (1973) and Lehr (1988); in North America we have: Dasypogoninae (11 genera), Stenopogoninae (31 genera), and Trigonomiminae (= Trigomiminae of Lehr; 4 genera). *Nannocyrtopogon* and its allies are stenopogonines.

ronmental influence. Even were it so, to have responded to that external stimulus in the manner required, the fly's genotype necessarily included within its repertoire an otherwise unexpressed capacity to provide the biochemical and developmental prerequisites for production of a nearly unblemished wing with that particular pattern, as later explained. If an external influence was involved, most probably it only indirectly instigated the necessary genotypic response (see below).

Among many conceivable genetic explanations, two well-known sporadic events may equally well account for the patterned wing. Because male asilids of known karyotype are either XY or XO, and females XX (Makino 1951, Cooper, unpublished), the fact that the chimaera is a male places a different restraint on the nature of the chromosome involved in each case. These events are:

1) Somatic mutation of a gene in the differential segment of a *sex chromosome* (+s/o → s/o),[5] the new allele's recessive phenotype therefore being expressible in its present hemizygous state. The mutation may have arisen by action of an external agent (e.g. by environmental radiation, mutagens, etc.), or internally (by replicative error, transposon, etc.).
2) Somatic crossing-over (see Stern 1968) in an *autosomal* heterozygote for an allele (a) giving a recessive phenotype, namely (+a/a → a/a and +a/+a equally). Studies of such crossing-over in *Drosophila* led Stern (1936) to conclude that it may in fact prove the most likely cause of somatic mosaicism *when* suitable heterozygosity is present.

If the frequency of the allele (a) were as high (but no higher) than 0.17 in the population of *N. minutus*, more than 160 flies (a number considerably larger than that of the reported and probable specimens now in collections) would be required for a 99% likelihood that at least one (a/a) individual with both wings patterned would be included within the sample. None has been reported, or described as a new species, as would be likely had such a specimen been found. The requisite heterozygotes (+a/a), however, would be relatively common (*ca.* 28% of both sexes).

In both cases the genetic change is assumed to involve an allele giving a recessive phenotype because most realized mutants with dominant phenotypes are far less common and more likely to produce malformations (catalog in Lindsley and Grell 1968). The change would necessarily occur in a nucleus at an early cleavage division of a preblastodermic egg. In that way a cell of a new genotype (either s/o or a/a) could have given rise to a sufficiently large clone to have formed the imaginal disc of the left wing, and perhaps other tissues of the chimaera. A mosaic arising from somatic crossing-over after the first "cleavage" division would be a trisectorial mosaic, in contrast to the bisectorial mosaic produced by a single somatic mutation.

Discussion

However the mosaic arose, it is clear that identical modes of genic action may be ascribed to the mutant allele, whether new (s) or preexistent (a). Choices for the results of such genic action in the case of the mosaic are two: (1) a discontinuous phenotypic change qualitatively different from that of wild type, a complex phenotype without precedent; in effect a preview of a potential apomorphy in the descendants of *N. minutus*; or (2) a recovery of an ancestral wing pattern, or nearly so; an atavistic expression which, were it found characteristic of a population today, would no doubt be viewed as an apomorphy.

Whether newly mutated or not, a struc-

[5] The "o" in these formulae indicates that there is no genic portion of the alternative sex chromosome, if any, that possesses the wild-type allele (+s), or a gene that suppresses the phenotypic action of (s).

tural gene does but one thing: it codes for the production of a single product. For many mutants, perhaps most, that product ultimately may play an active role in more than one biochemical pathway in development, giving rise to one or more seemingly unrelated phenotypic effects. Such an allele is said to be "pleiotropic" in its action. Thus *Lyra of *D. melanogaster* affects the eyes, body setae, wings, abdominal tergites and color; when homozygous it is lethal.

Of the thousand or more loci for which mutant alleles are now known in *D. melanogaster* (Lindsley and Grell 1968—"... reasonably complete through 1966"), most of the alleles have adverse pleiotropic effects. Alleles at nearly a third of the loci have an effect upon the wings, and about an eighth affect only the wings in one or more ways (catalog in Braver 1956). The phenotypic changes in the wings are almost always anomalous, among which are minor to extreme abnormalities of the blade, of its margins, of venation, of acanthae, of color, retention of hemolymph, and of expansion of the pupal wing at eclosion. Though many *Drosophila* species have maculated wings, including males of some members of the *melanogaster* subgenus (*Sophophora*) Bock and Wheeler (1972), none of the known phenotypes of male or female *D. melanogaster* take the form of a wing with a color *pattern.*

Those mutant alleles which do produce a new coloration of the blade without accompanying abnormalities of the wing are but a tiny minority of all; e.g. fuliginosus (Buzzati-Traverso 1947), *lemon, *pallid and *yellow. All such alleles at the four loci, except one (y^{50b25}, Gianotti 1951), affect both body and wing color in similar ways. Their primary effect is evidently upon the capacity of epidermal cells to produce particular melanins rather than an exclusive effect upon the epidermal cells of the wing itself. The latter appears to have been the case for y^{50b25}.

If the very extensive observations on *Drosophila* reflect in a general way attributes of mutations of flies, then comparable mutational changes affecting only coloration of the blade of the wing are expected to be extremely uncommon.

Compared with the mutant alleles that affect the wings of *D. melanogaster,* that presumed in *N. minutus* to have brought about maculation of the left wing is astonishing in the complexity of its phenotype and freedom from gross malformation. The phenotype leaves coloration of the small venational "cells" in the stalk of the wing (Fig. 4, "cells" 1–5) and most of the membrane of the blade unaffected (Figs. 1–3). However, it selectively heightens the levels of pigmentation, to very different degrees, in two unequally shaped, large, well-separated groups of contiguous epidermal cells (cf. Figs. 3 and 1, 2, 5). The pigmentation of the newly maculated areas is cell-produced and cell-limited; the boundaries between pigment cells and adjoining normally colored membrane are therefore sharply defined. Even venational cells r_1 and r_{2+3}, the bases and apices of which are of greatly different intensities of brown, show not the slightest signs of a decreasing color gradient from dark to light.

If all this resulted from a single product coded by a new allele, that product must have enhanced pigment formation (which awakens no problem) yet have benignly activated a series of coordinated pathways not ordinarily revealed by a difference in pigmentation basally and apically, nor by any partitioning of the wing into such special domains other than by veins. No mutations recorded for *D. melanogaster* produce *de novo* comparably complex, well-ordered phenotypes in any structure without notable abnormality. The circumstances appear to call for another interpretation of the origin of the pattern.

In perhaps most populations there is a phenotypically unexpressed retention of genetic bases for one or more ancestral attributes. Indeed Garcia-Bellido (1983) commented that "... it is not impossible ...

that most new patterns found in evolved groups of *Drosophila* are ancestral patterns."[6] In fact, Richards (1958) has demonstrated just such a case in *Ephestia*. Furthermore, Sondhi (1962), by continued selection in a strain of *D. melanogaster*, was able to produce a wholly new pair of bristles, in a particular location, comparable and presumably homologous with those found in the related family Aulacigastridae, and very probably with those in an ancestor of the two families. Causes and means for continuance of such apparently "silent" genic presences within the genome are discussed by Regal (1977), Riedl (1977), Hall (1984), and Coyne and Prout (1984) among others, along with examples from a variety of reactivated phenotypic expressions of such concealed bases of ancestral attributes otherwise known only from fossils. Gauld and Mound (1982) have discussed apparently frequent reversals and the problems they necessarily awaken in phyletic analysis.

To most there would seem to be an unbridgeable gap of complexity between most "ordinary" mutations and one that seems to call forth a probable attribute of an ancestor of countless generations removed. Is that so?

A mutant allele that restores the expression of an ancestral attribute does not differ from other mutant alleles with less striking phenotpes in kind, in degree, in mode of action in a developmental pathway, or even necessarily in the phyletic age of the pathway affected. It differs solely by its chance triggering and disclosure of a latent, ancient, yet still potentially expressible system within the genome. The difference is therefore not the nature of the mutation, but resides in a special peculiarity of the genome itself—a retained but suppressed integrated system, a "prepattern," in this case for wing maculation.

[6] I would add "perhaps most often in a somewhat modified form because of their reexpression within a changed genetic milieu."

The mutant allele codes for a product just as in other cases, but that product makes biochemically possible release and expression, wholly or in part, of the existant coordinated but "silent" ancestral pathways within the present genetic system. No chance pleiotropic concatenation of pathways to produce a coordinated wing pattern need be involved—that Achilles heel of the hypothesis of a wholly new phenotype. They already exist in a coordinate relation owing to prior evolution. The minor abnormalities expressed in the patterned wing of the mosaic may owe either to a pleiotropic effect of the mutant, or they may reflect a loosening of developmental timing within the retained ancestral system now being reexpressed against the milieu of new mutations accumulated since suppression of the ancestral wing pattern became a lineage attribute, or both. In any case, the reappearance of an ancestral wing pattern (or close thereto, perfect reversion being unlikely) seems to me the more plausible interpretation of the left wing of the mosaic *N. minutus*.

In the absence of evidence from fossils[7] of likely ancestral stocks, atavism cannot be disproven or proven for the wing of the mosaic. Nevertheless it does seem plausible because the complex maculated pattern in a nearly perfect wing of *N. minutus* appears otherwise as a freak of nature, for all 28 species of *Nannocyrtopogon* have either hyaline or lightly infuscated wings. However, as earlier mentioned certain species of

[7] The relevant Oligocene-Miocene fossils are assigned by their authors to stenopogonine genera contemporaneous with *Cyrtopogon* and *Nannocyrtopogon* (namely *Ceraturgus* [as *Ceraturgopsis*], 1 sp.; *Dioctria*, 2 spp.; *Holopogon*, 2 spp.; and *Microstylum*, 1 or 2 spp.). See Hull (1962) and Papavero (1973) for references and comment. So far as can be told all have either hyaline, not-maculated, or infumated wings. However, absence of maculations in a fossilized wing is not of itself reliable evidence for a corresponding absence of color pattern in the wing prior to fossilization (see Carpenter 1971).

the presumed sister group, *Cyrtopogon*, do have strongly maculated wings of interrelated patterns, some with both a central and apical macula. Those patterns are somewhat less complex, and the maculations differently shaped, defined, and placed than those of the left wing of the mosaic. Because *de novo* origin of such a complex pattern by a single mutation is highly improbable, and by simultaneous multiple mutations implausible, it is reasonble to assume that *Cyrtopogon* and *Nannocyrtopogon* shared an ancestor with a patterned wing, and that the means for wing patterning was retained in both lineages.[8] In the line from which *Nannocyrtopogon* species were derived, however, expression of pattern was suppressed. Retention of the suite of ancestral mutants involved presumably owes to their still essential contribution to one or more stages of development. Only their inessential actions, as those leading to an expression of a pattern, are genetically suppressed. The new mutant (s) or homozygote (a/a) then codes for a product of which the ultimate effect is reactivation of suppressed pattern pathways.

I now turn to another atavism, widespread among asilids and other Brachycera, that does not appear to have received the attention merited. Hennig (1954) raised the question as to whether the presence of vein R_3 in the asilid *Promachus* and its apocleine relatives represents an atavism. He thought not, although he left the question open for mydids and possibly others in which only the distal stub of R_3 or its trace remains. R_3 occurs also in the genus *Pseudorus* which is only remotely related to *Promachus* (Papavero 1973). Shannon and Bromley (1924) indicated the presence of R_3 in *Pogonosoma*, another asilid of rather remote affinity to both *Pseudorus* and *Promachus*, and in one or another form in other asilids, many bombyliids, some leptids, mydids, tabanids and occasionally in therevids. Very likely R_3 in these and perhaps other families is a vein tending to widespread reduction and loss at individually different rates throughout the Brachycera (in many families it has already been lost), a kind of "orthogenesis," much as appears to be happening to the basal length of M in flight wings of beetles.

The isolated, regular occurrence of a *complete* R_3 in certain species of *Pseudorus* (as Papavero 1973 suggests; see figs. 4, 5 in Oldroyd 1964), and in the 18 or so species of *Pogonosoma*, may represent recurrences rather than prolonged retention of R_3 beyond that of the numerous other members of their subfamilies. Certainly the well-known "anomalous" occurrences, most frequently asymmetrically, of a remnant of R_3 (as a "stump vein," = Tillyard's 1919 "interradial crossvein") or, more rarely, in complete form in individuals of species normally lacking all traces of R_3, are to be regarded as atavisms. Such individuals demonstrate that suppression of the R_3 phenotype is still not complete in their species, and that frequency and penetrance are low for the gene(s) still capable of restoring the phenotype. Unlike the maculated wing of the mosaic *N. minutus*, such cases do not require genetic mosaicism.

The general notion that the loss of an attribute in evolution, especially loss of a complex one, tends to be unrecoverable in later descendants was first proposed by Meyrick (1884), and apparently independently by Schlosser (1890), Gadow (1893), Dollo (1893) and, for plants, by Arber (1919). Such "lost" attributes, perhaps most of them, probably become unrecoverable in recognizable form only long after complete disappearance by genetic inactivation of their obvious phenotypic expression from a population. That is, only after potential co-

[8] Contrary to Meijere's (1907) opinion, ancestral flies, especially those of the Nematocera and of early Asilomorpha, probably did not have hyaline wings (all had epidermal "melanin"-producing pathways). Probably wing maculations of one sort or another were at least as common among them as they are among modern forms.

ordination of their genetic bases has finally been lost from the genome are they beyond mutational recall.

Loss of a phenotypic attribute and total loss of its recoverability have different immediate causes, and probably regularly occur stepwise over long but varying intervals of time. Just as that last known occurrence of an extinct form gives an unreliable date for *de facto* extinction (witness the coelacanth *Latimeria*), so also the time of final loss of recoverability of an apparently vanished attribute within a lineage must generally remain a matter of guesswork.

Conclusion

Though both somatic crossing-over and somatic mutation (or still less frequent genetic events) may formally account for the origin of the mosaic's patterned wing by reactivation of suppressed genetic pathways of distant ancestors, somatic mutation seems the simpler, more likely hypothesis. Both hypotheses predict certain possible outcomes by which they may be differentiated:

If the mosaicism was caused by somatic crossing-over in a fly of a population carrying an autosomal allele established at a moderate frequency, whether or not spermatogonial cells were included within the new (a/a) section, it is possible that a male or female will be found with both wings displaying the striking new color pattern in one or another population of *N. minutus*. No significant sexual difference in frequency would be expected were numbers of such flies found. Additionally, similar wing mosaics may turn up in the future because conditions for their formation are present in the population.

On the other hand, if a newly mutated sex-linked gene (s) were the cause of the mosaicism, no future finding of individuals with both wings maculated would be expected unless the mutant sector included at least some spermatogonial cells in addition to the left wing's imaginal disc. Even so there would be but a small likelihood of (s) entering and persisting in the local population at Holcomb Valley. It would depend on the male's success in leaving (s/+) female progeny, sampling error, and local population size. If (s) did persist in the population, no flies with maculated wings would be expected in the first filial generation. Thereafter males with maculated wings, though rare, would be greatly more frequent (about two orders of magnitude) than such females. If (s) did not persist in the population, reoccurrence of a similar wing mosaic, or of flies with patterned wings, would require a new mutation to the same allele or an isoallele.

Deposition of Specimen

For the present the specimen remains in my possession.

Acknowledgments

I am indebted to Dr. Eric M. Fisher (Calif. Dept. Agric.) for identifying the asilid. He and Profs. E. Gorton Linsley (U. Cal., Berkeley), John Pinto (U. Cal., Riverside), and my former colleague in genetics, Dean R. Parker, most helpfully commented on an earlier draft of the article. To my colleagues William L. Belser and Karl Fryxell I am indebted for their discussions of certain genetic implications awakened by the mosaic. Saul Frommer, curator of insect collections at U. Cal., Riverside, generously provided facilities for the study of the fine collection of Asilidae in his care. To Profs. Hampton L. Carson (U. Hawaii), Henry A. Hespenheide (U.C.L.A.), R. Lavigne (U. Wyoming), C. Riley Nelson (U. Utah), and Drs. Gregg J. Gunnell (Museum of Paleontology, U. Michigan) and Norman E. Woodley (U.S.N.M.), I am grateful for their cordial replies to my queries. None, of course, are responsible for the analysis presented.

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